ABSTRACT OF THE DISCLOSURE

The present study demonstrates that brimonidine tartrate, an alpha-2 adrenergic receptor agonist, can prevent photoreceptor cell degeneration and the associated Muller cell degenerative signs in an in vitro model of retinal degeneration and retinal detachment (separation of the neuroretina from the retinal pigment epithelium). Similar to control conditions, brimonidine allowed for the formation of highly structured photoreceptor outer segments, prevented the expression of stress markers in Müller cells and preserved the expression patterns of Muller cell markers of proper cell-cell contact and differentiation. Ultrastructural studies also indicated that brimonidine favored the formation of cell-cell junctions between photoreceptor cells and Müller cells, indicating that this phenomenon is associated with the exertion of the neuroprotective effect. The results suggest that brimonidine compounds may be utilized as an effective therapeutic agent for early and late onset retinal degenerations caused by defects in photoreceptor cells, Müller cells or both, and as an adjuvant to therapeutic success in retinal detachment surgery or macular translocation surgery for age-related macular degeneration.